REMARKS

Applicants respectfully request reconsideration of the maintained and newly presented rejections of the claims of the instant application in view of the amendments above and the following remarks.

I. STATUS OF THE CLAIMS

Upon entry of the foregoing amendments, claims 1-20 will be presently pending. Claims 1, 5, and 19 have been amended without prejudice. No claims have been cancelled. No new claims have been added. Claims 9-11, 13-15, and 18 have been withdrawn from consideration. Applicants respectfully submit that upon allowance of a generic claim, they may request rejoinder of claims directed to non-elected species written in dependent form or which in the alternative contain all limitations of the allowed generic claim. Applicants also reserve the right to file one or more additional continuing applications directed to any cancelled or non-elected subject matter.

Claim 1 has been amended to more clearly recite the subject matter of the present invention. Specifically, claim 1 now recites a method of inhibiting cancer development in pre-cancerous cells. Support for this amendment is provided in the original specification at least at page 6, lines 10-20; page 8, lines 6-13; and page 9, lines 10-12. Claim 5 has been amended to correct its dependency to claim 4. Claim 18 has been amended to clarify the subject matter recited therein by adding the word "wherein." No new matter has been added.

II. REJECTION UNDER 35 U.S.C. § 103(a)

In the present Office Action, claims 1-5, 8, 12, 16-17, and 19-20 have been rejected as allegedly obvious over Kuhajda et al.[1] in view of Wang et al.[2] Claims 6 and 7 have been rejected as allegedly obvious over Kuhajda et al. in view of Wang et al., further in view of Hirsch et al.[3] Claims 1-8, 12, 16-17, and 19-20 recite, among other aspects, a method of inhibiting cancer development in pre-cancerous cells comprising the administration of an effective amount of a fatty acid synthase (FAS) inhibitor.

[1] PNAS, 2000, 97(7):3450-3454.
[2] Zhonghua Zhong Liu Za Zhi, 2002, 24(3):271-273.
[3] British J. Cancer, 2002, 86:1449-1456.

Kuhajda et al. is directed to the synthesis of C75 and antitumor activity as an inhibitor of fatty acid synthase. Wang et al. relates to the expression of FAS in non-small cell lung cancer (NSCLC). Hirsch et al. is directed to the significance of HER-2/*neu* protein expression in NSCLC. While the Examiner acknowledges that Kuhajda et al. does not specifically teach a method of inhibiting lung cancer utilizing C75, the Examiner states that it would have been obvious to do so in human patients who also express *neu* protein because Wang et al. discloses that FAS expression was detected in human NSCLC patients, and Hirsch et al. discloses that NSCLC patients express the *neu* protein. Applicants respectfully traverse this rejection at least because Kuhajda et al. does not teach or suggest inhibiting cancer development in ***pre*-cancerous cells**.

Applicants are more than familiar with Kuhajda et al., as Dr. Kuhajda is the first author of the reference and the first named inventor of the instant application. Applicants agree with the Examiner that Kuhajda et al. discloses that C75 binds to and inhibits FAS in human **cancer cells**. See Office Action, p. 5; see also Kuhajda et al., p. 3450, abstract and right column. Kuhajda et al. does not disclose, however, a method of inhibiting cancer development in ***pre*-cancerous cells**; neither does Kuhajda et al. contain any speculative language suggesting the same. Kuhajda et al. observes that C75 may enable the study of FAS inhibition "in human cancer." Kuhajda et al., p. 3450, right column. To this end, Kuhajda et al. discloses the effects of FAS inhibitors on pre-existing cancer cells, e.g., SKBR3 breast cancer cells and HL60 human promyelocytic leukemia cells. Thus, while it was known that C75 can ameliorate pre-existing cancer cell growth, it was not known that treatment with FAS inhibitors would inhibit cancer development in pre-cancerous cells. See Orig. Spec., p. 17, lns. 34-37.

To this end, the instant invention provides "a method for treating the ***pre*-cancerous state** in a subject (i.e., inhibiting cancer development)." Id. at p. 6, lns. 10-12 (emphasis added). The specification as filed defines the term "inhibiting" as "delaying cancer development . . . by stimulating, inducing or triggering apoptosis . . . in ***pre*-cancerous cells**." Id. at p. 8, lns. 6-9 (emphasis added). The specification further defines "cancer development" as "the initial appearance of cancerous cells," i.e., "cells which have the property of autonomous proliferation and have invaded adjacent tissues." Id. at p. 8, lns. 10-13. In this regard, a method of inhibiting cancerous development in pre-cancerous cells is significantly distinguished from a method of treating the growth of pre-existing cancerous tumor cells.

Examples 4 and 5 in the specification of the present application demonstrate quite clearly that FAS inhibitors inhibit cancer development in *pre*-cancerous cells, as distinguished from treating existing cancerous cells. To show that FAS inhibitors inhibit cancer development, the HER-2/*neu* breast cancer transgenic mouse model is used to show inhibition of cancer development. *neu*-N transgenic mice express the non-transforming rat *neu* cDNA under the control of a mammary-specific promoter. As a consequence, the mice develop spontaneous mammary adenocarcinomas beginning at approximately 125 days, with nearly all of the mice harboring tumors by 300 days. In this regard, the HER-2/*neu* transgenic mouse model may be used to evaluate test compounds for effectiveness as inhibitors of cancer development. Specifically, a test compound would be administered to a test group and withheld from a control group. Suppression or delay of cancer development in the test group relative to the control group would indicate the effectiveness of the test compound as an inhibitor of cancer development.

This is the exact result demonstrated by Example 4. In Example 4, Applicants monitored the development of cancer in thirty HER-2/*neu* breast cancer transgenic mice. Fifteen of the mice received weekly doses of C75, a FAS inhibitor, for three months beginning at five weeks of age. No FAS inhibitors were administered to the remaining fifteen mice. As Applicants disclose, 50% of the control mice developed tumors after approximately 200 days versus 300 days for the animals treated with a FAS inhibitor. Significantly, three treated animals remained tumor free for nearly 18 months.

Kuhajda et al. does not disclose nor suggest the method of inhibiting cancer development in pre-cancerous cells by the administration of a FAS inhibitor, as demonstrated by Examples 4 and 5. Absent the disclosure of the instant invention, there is no teaching in the art that using FAS inhibitors on pre-cancerous cells would work to inhibit cancer development. Thus, Kuhajda et al. does not render the claimed invention unpatentable, and the Examiner is respectfully requested to withdraw these rejections.

CONCLUSION

In view of the abovementioned amendments and remarks, Applicants respectfully assert that this application is now in condition for allowance. The Examiner is invited to contact the undersigned counsel in order to further the prosecution of this application in any way.

Respectfully submitted,

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